Ranney (A. L.)

Compliments of the Author.

CAUSES OF DEATH

IN

SURGICAL OPERATIONS.

BY

A. L. RANNEY, M.D.,

Adjunct Professor of Anatomy in the Medical Department of the University of the City of New York.

Reprinted from THE MEDICAL RECORD,
June 28, 1879.

NEW YORK:
WILLIAM WOOD & COMPANY,
27 GREAT JONES STREET.
1879.

CAUSES OF DEATH

IN

SURGICAL OPERATIONS.

BY

A. L. RANNEY, M.D.,

Adjunct Professor of Anatomy in the Medical Department of the University of the City of New York.

Reprinted from THE MEDICAL RECORD,
June 28, 1879.

NEW YORK:
WILLIAM WOOD & COMPANY,
27 GREAT JONES STREET.
1879.

CAUSES OF DEATH IN

SURGICAL OPERATIONS.

It is my desire in this paper to direct attention to those causes of death which occur during surgical operations, and to study those measures on the part of the surgeon which will tend to modify their severity, or, by preventing their occurrence, decrease the danger to life.

I shall divide these causes of death into: first, those dependent on the *blood-vessels;* second, those dependent on the *nervous system;* and, third, those dependent on the *blood.*

I shall consider, under the first, causes dependent on vessels: 1st. Hemorrhage; 2d. Air in the veins; 3d. Apoplexy; 4th. Embolism.

Under the second, causes dependent on nervous system: 1st. Shock; 2d. Collapse.

Under the third, causes dependent on blood: Anæsthetics.

I proceed first to the discussion of hemorrhage, as the most frequent cause of death during surgical operations.

HEMORRHAGE.

To die of repeated hemorrhages, while perhaps the most painless of deaths, is the most awful. With each succeeding hemorrhage, dissolution is so distinctly intimated, and the patient is so conscious that his strength is fast ebbing, that he clings to life. The most resolute are overcome with an anxiety which they cannot conceal, and look around for some one to delay, at least, the fatal moment.

But if there be a *sudden* hemorrhage from a vessel opened in an operation, or from an aneurism, or from some wound, the arteries of which cannot be discovered, there is *immediate* danger of death to the

patient, even while in the hands of the surgeon. Those who have witnessed the agitation of such scenes can best judge of the importance of this subject.

John Bell, in his essay on hemorrhage, though undoubtedly influenced by the defective means of arrest then existing, closes a paragraph with these words: "Were this *one* danger removed, would not the young surgeon go forward in his profession almost without fear?"

I question if even our present enlightenment has entirely dispersed this element of anxiety.

Hemorrhage is usually classed into three distinct varieties: 1st. *Arterial*, as indicated by a bright red color of the blood and an intermitting flow; 2d. *Venous*, as indicated by a continued flow and darker color: 3d. *Parenchymatous*, as indicated by a *general* oozing from the capillaries of injured surface.

CAUSES PRIMARILY MODIFYING HEMORRHAGE.

The amount of hemorrhage is *modified* at first by the number of vessels wounded and by the smoothness of the *cut* surface.

This latter element is especially important in vessels of large calibre, since an injury with a dull instrument, or the rending of a vessel from violence, leaves the mouth in a condition to offer more or less resistance in itself to the rapid escape of blood, independent of the changes which occur in the coats of the wounded vessel subsequent to its injury.

CAUSES TENDING TO PROLONG HEMORRHAGE.

There are, however, other causes which may modify hemorrhage *later on*, and, by *prolonging* it, tend to greatly increase the danger to life. Under this head I would mention:

1st. *Gravity.*

2d. *High temperature*, whether in the wound or surrounding atmosphere, by delaying coagulation.

3d. *Muscular expiratory efforts*, especially in wounds of the neck.

4th. *Obstruction to a free venous return.*

5th. *Obstructed or delayed contraction of vessels*, as occurring in hepatic hemorrhage; from the teeth;

from the nutrient artery of bone; from disease of the vessel; and from atony of vessel.

6th. *Diseases of blood*, preventing coagulation or assisting exudation, as in *vicarious hemorrhage.*

7th. *Congenital anatomical defects* in the construction of vessels. Wilson's case: *Lancet*, 1840; coats only one-half normal thickness. Blagden's case: *Med. Chir. Trans.*, vol. viii., p. 224; transparent coats—died from pulling tooth. Trachsmuth's case—death from ruptured hymen.

PATHOLOGICAL CHANGES DEPENDENT ON HEMORRHAGE.

These changes may properly be grouped under three heads:

1st. Changes in the constituents of the blood.

2d. Changes in the heart's action.

3d. Changes effected in the tissues.

There has been much discussion whether changes in the blood constituents, dependent on injury to tissues, and vessels, are always uniform. I think that in those forms of violence causing marked injury to tissues, that all the steps of an inflammatory process must necessarily occur within the injured tissues, and that the customary blood-changes in inflammatory blood must also *coexist*, provided that sufficient *irritation* has been produced to cause constitutional as well as local symptoms.

But I am by no means certain that in those cases of injury to a vessel, where the tissues are but *slightly* involved and the hemorrhage small in quantity (thus causing little, if any, direct constitutional effect), a markedly altered *blood-condition* is developed, or that increase of the fibrin and albumen could well be verified.

I am inclined to regard "blood-changes," dependent on injury to vessels and tissues, as but a slight factor in the reparative process which nature sets up unaided; and also to think that the "increased plasticity" of the blood, so much discussed as aiding nature's arrest of hemorrhage, is not only dependent on the amount of irritation produced by the injury on the tissues, but in many cases may be wholly absent.

Not so, however, with those changes in the blood

localized within the wound, and dependent, not on chemical alteration in its constituents, but rather on mechanical obstruction in the vessel, from the increased *density* of its fluid, resulting from a sluggish current and constant exudation of the plasma.

I do not feel that a lengthy description of the changes in the vessels following an irritant is admissible; but we have, doubtless, all perceived how in the web of a frog's foot, under the glass, are seen, successively, the stage of capillary contraction, of capillary dilatation, resulting in active hyperæmia, and followed subsequently by congestion, stasis, swelling of tissues from exudation of plasma elements, and transudation of white corpuscles.

We know, also, that the red blood-corpuscles, when deprived of the normal amount of plasma by exudation, show an *apparent* increase of adhesiveness and a tendency to pack themselves together like *coin* in bundles.

This *adhesiveness* seems to me, however, a purely local condition and a mechanical result of the escape of the plasma, not attributable to a general blood change or necessarily a *prima facie* evidence of general increase in the fibrin.

We may state then, in summary, without going too deeply into argument, that the changes in the blood, dependent on injury to vessels, are of a *local* and *constitutional* character; that the local conditions are mechanical in their causation, and, therefore, more or less uniform. While the constitutional conditions are dependent on some changes, resulting from irritation at the seat of injury, and are variable, not only in their degree, but even in their actual existence.

We pass to the second set of changes, due to injury of the vessel, viz., "changes occurring in the heart."

II. *Changes in the Heart.*—Immediately upon the opening of a vessel of large calibre constitutional effects appear in direct proportion to the amount of blood lost. These effects are mostly confined to the heart and general circulation. The heart gradually loses its normal power, and becomes accelerated in its action. The arterial tension in the superficial vessels shows rapid diminution, and the pulse changes in volume from its normal character, becoming smaller

and even *thready* in cases where the hemorrhage has been alarming.

III. *Changes in the Tissues.*—The third class of pathological conditions, viz., "changes in *tissues*, dependent on injury to vessels," is found to exist in two distinct situations: 1. In coats of the artery themselves; 2. In the tissues about the artery.

When an artery of small size is wounded, we notice frequently that without any form of treatment, not even compression for a short period, the hemorrhage ceases spontaneously and within a very short time. We notice, in the second place, that this spontaneous arrest of hemorrhage is more rapid in some anatomical situations than in others; and, in the third place, that this spontaneous arrest of hemorrhage depends somewhat on the variety and extent of the injury.

To explain the conditions to which these variations are due, and to positively reach the exact changes in tissues on which this spontaneous arrest of hemorrhage depends, has for centuries been a subject of investigation, inquiry, and dispute.

Petit, in 1731, claimed that the arrest was due not to contraction of the vessel, but to the formation of two clots—an outside clot called "*couvercle*," and an inside clot called "*bouchon*."

Morand, in 1736, added changes in the arterial coats.

Kirkland, in 1763, added the effect of decreased heart's action and sustained arterial contraction, but denied the influence of coagula.

John Bell denied contraction and *internal* coagula as a means of arrest, but advocated "infiltration of blood into the cellular tissue."

Jones, in 1805, in his wonderful essay on hemorrhage, by experiments, advanced doctrines which to this day have been little altered.

These changes, as now accepted, are as follows:

1st. A retraction of the whole vessel within its sheath occurs, due to its normal elasticity.

2d. A contraction of the entire coats of the vessel in some cases ensues, causing a conical appearance of the severed end, or, in others, a *curling* of the middle and internal coats into the calibre of the injured vessel is perceived.

3d. The formation of an internal and external coagulum is usually detected, the latter being, however, possibly absent.

4th. "Adhesive inflammation" is now excited between the clot and internal coat, also between the three distinct arterial coats, and often between the external coat and the outside tissues.

5th. Organization of the internal coagulum, with development of blood-vessels within it, and a free anastomosis between them and the neighboring capillaries, completes the process of repair.

Guthrie states that in the *distal* end of an injured vessel these changes are, as a rule, imperfectly performed; that arterial contraction is deficient, and that, for that reason, secondary hemorrhage from the distal end is most common.

A much better explanation of this latter occurrence, to my mind, is based on an anatomical alteration between the proximal and distal extremities of an injured vessel, since, in the latter, the vaso-motor nerves are frequently severed from their direct *ganglionic* attachment; and from *defective nervous* influence, reparative processes are either delayed or imperfectly performed.

SYMPTOMS DUE TO HEMORRHAGE.

When a patient expires suddenly from the impetuous bleeding of some large artery, from a ruptured aneurism or wounded viscera, the face at once becomes deadly pale, a dark circle round the eyes is perceived, the lips change to a blackish hue, and the extremities become rapidly cold. The patient faints, revives but to be conscious of his danger, and faints again. The voice is lost; there is an anxious and incessant tossing of the arms, with that restlessness which is the sign of the approaching end. The head is suddenly raised, gasping as it were for breath, with inexpressible anxiety depicted on the countenance. The tossing of the limbs continues; convulsive sighs are drawn; the pulse flutters, intermits from time to time, and the patient expires.

The countenance is not of a transparent paleness, but of that clayey and leaden color which the painter represents in assassinations and battles; and this toss-

ing of the limbs, which is commonly represented as the sign of a fatal wound, is indeed so infallible a sign of death, that I have never known any one to recover who had fallen into this condition.

Treatment.—In the early centuries, when hemorrhage was with difficulty controlled, and the percentage of mortality from this source enormous, superstition frequently accompanied the defective surgical means at that time in vogue. Thus we find Wolfius, Saurtus, Michael Mercates, and Gottfried Macbius, in the sixteenth century, extolling the application of TOADS behind the ear and in the arm-pits, as a means of arresting hemorrhage.

Plunging bleeding members into the abdomen of a living fowl had its adherents. The use of hot magnetic ore, boiling oil of turpentine, vitriol, and corrosive sublimate were also among the cruel practices of the day. The actual cautery can be found described as early as Galen. Albucasis, in his work on surgery, devotes fifty eight chapters to the cautery and its uses. All possible designs and shapes were wrought from iron to meet the various emergencies, and plates of them published, and the special advantages of each extolled. Red-hot knives were first suggested by "Fabricius Ab Aquapendente" as a valuable improvement on former customs for the *immediate* arrest of hemorrhages during amputation.

In the reign of Henry IV., Ambrose Paré first advocated ligature, with rules and directions not unlike those of the present day; but for a century it was used with great caution, and met with much disfavor.

Petit, in 1730, urged a compress and bandage at the stump to modify the *shape of the clot*, and invented the tourniquet, known by his name. In 1732 Petit's experiments of the effect of astringents on mutton were made in his endeavor to discover artificial means to *harden* the clot within a stump by local applications.

Pouteau, soon after, advocated the ligature of *nerves* with the vessel to stimulate the swelling of tissues, and thus cause compression of the vessel. Subsequently torsion became developed by Amussat, Velpeau, and Thierry. Ligature and its mechanism have been fully explained by Jones. Tourniquets have been modified and improved by Signorini, Skey, and hosts of others,

and the study of collateral circulation investigated to a high degree of perfection by Mannoir, Porta, and Stilling.

Transfusion has also been added, of the literature of which Blundell's Essay probably best deserves mention. Acupressure, devised by Simpson in 1859, has proved also a valuable contribution to this branch of surgical investigation.

To recapitulate, then, we have as surgical means of arrest:

1st. *Tourniquet* (a temporary measure): Petit's, Signorini's, Skey's Horse-shoe, and others.

2d. *Styptics:* 1. Cold used in cavities or to allay general oozing. 2. Ferri persulph. 3. Gallic acid. 4. Matico. 5. Alum. 6. Argent. nitratis.

3d. *Ligature:* Paré, Jones.

4th. *Torsion:* Galen, Amussat, Velpeau, Thierry. It is now decided that four complete turns are required to occlude the calibre of a vessel.

5th. *Acupressure.*

6th. *Cauterization.*

7th. *Transfusion of blood.*

I close the subject of hemorrhage by enumerating certain general rules of treatment, which seem to me to meet all possible indications.

1st. Always *ligate* the bleeding vessel, in moderate hemorrhage, when convenient to do so; the form of ligature used depending on the choice of the operator.

2d. Use compression over the wound on the *main trunk*, in moderate hemorrhage, when ligature of the wounded artery is inconvenient.

3d. In *violent* hemorrhage enlarge the wound and tie the artery.

4th. As a *rule*, never attempt ligation except when bleeding *actually exists.*

The exceptions to this rule are: 1. In *exposed* vessels of large calibre demanding ligature as a safety measure. 2. In delirium tremens following an injury. 3. When necessity for transportation exists.

5th. Ligature should, as a rule, be applied *at the bleeding point*, and not remote from it.

The reasons for this general statement being: 1. That collateral circulation may otherwise keep up the hemorrhage. 2. The bleeding vessel may not be the

main trunk. 3. There exists in certain localities additional danger as you approach the heart. 4. Gangrene is liable to occur, in case subsequent ligature of the wound shall be required.

6th. Use the *external wound* as a *guide* to your incision to reach the vessel—except when the wound exists on the side opposite to the vessel injured, when a probe may be cut down upon.

7th. Always use the greatest precaution to avoid needless loss of blood in reaching the vessel, until the finger can compress it.

8th. The artery, when found, should be tied above and below the wounded portion, and at a bifurcation THREE ligatures should be used.

In case the lower end cannot be discovered, use *compression* in the wound as a substitute for ligature.

9th. A ligature should not be placed close below a large branch.

10th. In *recurring* hemorrhages the treatment should depend on the *color* of the blood and on the severity of the hemorrhage.

If the hemorrhage springs from the proximal end of the artery: 1. Tie, if possible. 2. Amputate, if necessary. 3. Use styptics and compression, if both are impossible.

11th. *Amputation* is preferable to ligature: 1. When great swelling of the limb renders ligation difficult. 2. When exhaustion of the patient forbids further search for the vessel. 3. When *competent assistance* is needed and not attainable.

12th. In case a large vessel is injured, without actual hemorrhage, heat and flannels to the limb are indicated as a *preventive* measure.

13th. In case an aneurism is the seat of hemorrhage—provided the aneurism is *traumatic* in its origin—it should be treated on the same principles as if it were a wounded artery.

AIR IN THE VEINS.

Etiology.—This distressing accident has always occurred, as far as my research has extended, in operations about the head, neck, or axilla.

Experiments on animals—by Morgagni, Valsalva,

Bichat, and Nysten—have shown death to arise in the dog from an injection of three cubic inches of air into the jugular vein, and in the horse from three ordinary human expirations.

Bichat, in his experiments, concluded that even *one bubble* might result in death; but his associate investigators—Bell, Magendie, Amussat, Cormack, Wattman, Nysten, and Erichsen—do not verify such a conclusion.

Death seems to depend not only upon the amount of air introduced, but the rapidity of the injection seems also greatly to influence it.

In surgery, however, we have only to deal with the *spontaneous* admission of air into venous cavities, dependent upon a thoracic vacuum existing both within the pleuritic and pericardial sacs, and occurring during the act of inspiration.

Pathology.—There has been much discussion and many theories as to the exact physiological condition produced by the entrance of air into veins and the mechanism of its action as a cause of death.

Bell's theory argued that death was the direct result of *air upon the medulla oblongata.*

Cormack ascribes death to *distention of the right heart* alone, without abnormal valvular or pulmonary conditions.

Erichsen denies both of these conclusions, and claims that death results from *obstruction in the pulmonary capillaries* from the frothy condition of the blood, which resists the vis-a-tergo of the heart.

This theory has had great support; is based upon extensive experiments and pathological research; and, where small quantities of air enter veins, probably is correct.

Moore, however, in his paper on this subject, advocates an explanation, in which the *valvular* element is brought out as the most important factor, and which certainly merits attention.

He states his explanation briefly as follows: 1. Air, on entering the right ventricle, fails to close the wet tricuspid valve during the ventricular systole, from its light density and compressibility. 2. During the following diastole of the heart the air enters, or rather floats, into the pulmonary artery. 3. During the

second ventricular systole the wet *pulmonary* valves also fail to close, and adhere to the sides of the vessel. 4. The succeeding diastole now draws into the ventricle blood from both the auricle and the lungs. 5. The cardiac systole now returns the blood again to its original situation, as both the tricuspid and pulmonary valves are open, and thus the circulation becomes arrested.

These two latter explanations probably cover the mechanism of death; the former being the most plausible when the amount of air in the heart cavities is small, the latter when a large quantity of air suddenly is introduced.

History.—In 1818 Beauchesne reported the first case of this accident occurring during the removal of a tumor of the right shoulder, the internal jugular vein being wounded. The patient lived fifteen minutes.

Subsequently cases were reported by Dupuytren, Delpech, Castara, Roux, Ulrick, Mirault, Warren, Mott, Malgaigne, Bégin, Erichsen, Cooper, Clemot, and others. Some of those recovered; some met almost instant death; some died of pneumonia from bronchial irritation at a later date.

In all of these cases, however, an abnormal condition of the opened vein existed, characterized by a failure to collapse, termed by the French *canalization*.

This abnormal condition may be produced either *artificially* or by some *anatomical* changes.

A. *Artificial canalization* may result from one of four conditions, viz.: 1. Tension of the aponeuroses, holding the mouth of a vein open. 2. Veins opened by platysma contraction. 3. Traction on the pedicle of a tumor. 4. Vein at an angle of the wound opened by traction on the flaps.

B. *The abnormal anatomical conditions* producing canalization are: 1. Indurated cellular investments about the vein. 2. Induration, or inflammatory thickening of the venous coats.

Thus in Beauchesne's case traction existed; in Delpech's, hypertrophy of axillary vein; in Castara's, section occurred during traction; in Roux's, section occurred during traction; in Ulrick's, the vein was enclosed in a tumor; in Mirault's, the vein was enclosed in a tumor; in Warren's, tension from

position of arm existed; in Mott's, the *facial vein* was tense from position of the head; in Malgaigne's, the vein was enclosed in the tumor; in Bégin's, the jugular vein was tense from traction.

Symptoms.—When air is allowed to enter into the veins of a dog by section, there occurs: 1. A hissing noise, with gurgling at the mouth of the vein. 2. Struggles during the subsequent inspirations. 3. A churning noise at the apex of the heart during the ventricular systole, with a bubbling, thrilling, and rasping sensation on palpation. 4. The circulation becomes feeble, but the heart's action *remains forcible*. 5. The animal becomes unable to stand, rolls over, utters a few plaintive cries, is convulsed, extrudes its fæces and urine, and dies.

In man, however, there are additional symptoms given us by the *expressions* of the patient; thus the patient experiences terrible constriction in the thorax immediately after the air enters; screams, moans, and subsequently whines as the symptoms increase. The pulse early becomes imperceptible, the heart's action labored; convulsions come on rapidly, and death usually occurs. Still, in Mirault's case three or four hours elapsed before death; in Clemot's, several hours; in Beauchesne's, fifteen minutes; in Roux's and Malgaigne's, death from pneumonia ensued; while in Erichsen's and Cooper's cases, recovery took place.

Treatment.—As *prevention* of this accident is of vital importance, the following suggestions may be of value: 1. Always close the mouth of any *open* vessel *instantly*, both on the proximal and distal end, and compress every vein before cutting it. 2. Avoid raising *any tumor* on the *shoulder* in operations about the neck, head, or axilla, without protecting the large veins by pressure. 3. Compress, between the wound and thorax, if fear exists, during alterations in the position of patient or tumor. 4. Bandage the chest and abdomen tightly to prevent *gasping respirations*, which tend greatly towards this accident.

As to the plan of *actual treatment*, after the accident has occurred, many suggestions have been offered, though their value will depend somewhat on the views held as to the mechanism of death. Thus Moore advises a *supine* position, to allow the blood

to fall to the *back* part of the heart, and thus raise the tricuspid valves. Others advise the head low and the feet high, to relieve the anæmia of the medulla. Mercier suggests *compression of the femorals*, axillaries, and abdominal aorta, for the same object; but this is objected to by others on account of the necessity of venous return, which is retarded by this method. Warren advises either *bleeding* from the temporal artery, *tracheotomy*, or *stimulants*, as the indications seem to demand, with galvanic shocks across the chest in case the heart's action seems to fail. Gerdy *compresses the chest*, hoping to expel or facilitate the passage of air through the lungs. In apparently fatal cases, Amussat and Blandin recommend *suction of air* from the heart, by means of a catheter passed into the open vein, or into right jugular if the former be impossible, with compression of chest at the same time. Magendie and Roux advocate suction alone. Reid and Cormack suggest the *opening of the right jugular vein* to relieve the right auricle.

Many of these plans have to me serious objections. Compression of the chest, after the accident has occurred, seems useless, and increases the pulmonary obstruction. Bleeding from the temporal artery depletes the already empty arteries. Tracheotomy only relieves a secondary symptom (dyspnœa). Erichsen lays great stress on artificial inflation of the lungs to overcome the obstruction in the pulmonary capillaries, and suggests that mechanical respiration be kept up after this procedure, using at the same time ammonia to the nostrils.

Artificial respirations, with the *mouth* and *nostrils closed*, have been suggested as a remedial measure, the object being to expel air from the heart by the vein through which it entered. Finally, *injections of warm water* into the heart cavity, to render the valves movable, and subsequent artificial respiration to keep up the cardiac action, is resorted to, and recommended by Moore as a remedy in the severe type of cases. His steps for this operation are as follows: 1. Raise head during the injection, to allow air to escape through the fluid. 2. Open some vein in the neck, and evacuate its blood, to further assist the escape of

air. 3. Avoid throwing in additional air with the syringe. 4. Inject with force enough to FILL, but not *distend* the heart cavities. 5. To inject two ounces at a time, with velocity enough to raise the wet valves which are adherent to the walls of the heart. 6. To stimulate the heart's action, during the operation, by galvanism and artificial respiration.

Without pausing to elaborate these means of treatment, I pass now to the *third* source of death dependent on vessels, viz.: embolism and apoplexy.

EMBOLISM AND APOPLEXY.

By embolism we mean a plug in a vessel interfering with the circulation of the blood, and formed at a point more or less distant from the seat of obstruction; by apoplexy, the escape of blood from a vessel into the cerebral tissue. I have coupled the two, since *cerebral* embolism is the most common form in fatal cases, and because there are some marked points of similarity in the symptoms of *this form* and that of apoplectic extravasation. Both, if occurring during a surgical operation, have been preceded by abnormal anatomical conditions, and owe their occurrence to the excitement of the operation, rather than to any act of the surgeon. Still, as these pathological conditions which previously exist may be determined, or suspected, before the date of the operation, the occurrence of either of these causes of death is not an absolutely unavoidable accident, and, in many cases, not undeserving of censure.

Etiology.—Embolism is usually caused by a former endocarditis, producing vegetations upon the valves, though it may result from a phlebitis, or from spontaneous coagulation of blood in small capillaries, from pressure of tumors, a pyæmic condition, and other causes; these plugs subsequently becoming movable. The former condition, viz., vegetations on the valves, is, however, the one most likely to produce abnormal *cerebral* conditions, as emboli from the venous system are seldom arrested till they pass through the right heart, and are mostly entrapped in the pulmonary organs.

Apoplexy, on the contrary, is not necessarily associated with heart lesions, but rather is produced by an

atheromatous condition of the vessels, which exists as a result of a previous endarteritis from irritation of alcoholic blood and high living.

Symptoms.—The *common* symptoms of both of these conditions, when present, leading to error in diagnosis or treatment are chiefly: 1. Possible coma. 2. Paralysis. 3. Sudden death.

The *distinctive* symptoms between the two diseases, provided the attack is not immediately fatal, are best grouped as a differential diagnosis.

Apoplexy.	*Embolism.*
The attack, if serious, but not immediately fatal, is accompanied by *coma* and *insensibility.*	Under the same conditions *consciousness* is liable to be present during the attack.
The indications of *cerebral compression* are present, as shown by the following symptoms:	The indications of *cerebral anæmia* exist, as shown by the following symptoms:
The breathing is *stertorous.*	The respirations are normal (as a rule).
The face is *flushed.*	The face is *pale.*
The pulse is *full* and *slow.*	The pulse is *rapid* and *feeble.*
The pupils are irregular.	The pupils are uniform.
No aphasia exists (as a rule).	*Aphasia* is diagnostic.
The paralysis is *slow* in improvement.	The paralysis usually improves slightly within *twenty-four hours.*
The arteries are often felt to be atheromatous.	The arteries are normal.
No cardiac lesion exists if the attack be uncomplicated.	The aortic and mitral valves are usually found to be abnormal.
A history of previous *high living* is usually present.	A history of previous *rheumatism* and *endocardial inflammation* is generally detected.

Both may have been preceded by similar attacks; but in each a recurrence is liable to take place, even should no previous history of a former attack exist.

Treatment.—In severe cases of either of these conditions little can be done to relieve, save a symptomatic line of treatment. Cold to the head, venesection, and catharsis are usually employed in apoplexy, but more for the object of preventing further escape of blood than as curative measures. In embolism some improvement usually appears within twenty-four

hours, without treatment, from a supply of blood, through the collateral circulation; but the prognosis depends greatly on the situation of the embolus, its size, and its character. Fatty degeneration often removes the foreign body before degeneration of the brain occurs; and, in these cases, complete recovery may result.

We come now to the SECOND CLASS of causes of death, viz., "causes affecting the nervous system."

SHOCK AND COLLAPSE.

By the term "shock" is meant a state of body dependent upon a sudden or violent impression affecting some portion of the nervous system, and, *through a nerve-centre*, acting upon the heart. By "collapse," a state of *extreme shock* verging upon dissolution, but not resulting in immediate death. Life may be destroyed without pathological lesions, as in cases of sudden or violent blows upon the epigastrium, and as a result of powerful mental emotion; but these cases are uncommon, and are, as a rule, always preceded by one of the above-named conditions. It is to these two conditions (viz., shock and collapse), therefore, that I direct your attention as a cause of death during surgical operations.

Syncope is not to be confounded with collapse or shock, since it differs from them in three respects: 1. In its duration and degree. 2. In the mental condition of the patient and the acute sensibility of the special senses. 3. In its more rapid *crisis*, but less profound effects.

Collapse is divided, according to its symptoms, into three stages: 1. Stage of shock. 2. Stage of reaction. 3. Stage of excessive reaction.

This third stage has been described by Hunter as the "stage of irritability;" by some as the "stage of delirium;" by others as the "stage of inflammation;" and by Travers as the "stage of prostration with excitement."

Etiology.—The causes of collapse may be classified into

1st. *Injuries* involving a large amount of tissue or nerve trunks: Lacerations, burns, injuries to joints, injuries to organs, especially liver, testicle.

2d. *Poisons*, depressing the nervous condition: Tobacco, aconite, drastic cathartics, etc.

3d. *Shock to nerve-centres* resulting from blow on epigastrium, lightning, concussion of brain, mental emotions, excessive pain, sudden cessation of pain, cold douche.

4th. *Sudden* and *severe hemorrhage.*

5th. *Exhaustion*, as produced by excessive fatigue, excessive privation from food, prolonged suppuration, prolonged mental excitement.

There are also certain conditions which are predisposing towards collapse, such as "old age," "debility," prolonged anxiety, excessive fear, etc.

Pathology.—In fatal cases of collapse the pathological conditions are confined to the heart and circulatory apparatus; thus the heart cavities are usually found to be markedly distended; the right heart is engorged with blood; marked engorgements of the great veins and cavas exist; and imperfect coagulation of blood is present in the heart and large vessels.

Symptoms.—The symptoms of collapse differ with the various stages, and will be therefore separately enumerated.

The symptoms of the first stage (stage of *shock*) are: 1. Pallor of the whole surface of the body. 2. Bloodless lips. 3. A clammy moisture of the skin with drops of sweat on the forehead. 4. A countenance dull, shrunken, and contracted. 5. An eye deficient in lustre, and partially covered with the drooping lid. 6. Dilated nostrils. 7. A temperature depressed below normal. 8. Extreme muscular debility. 9. A pulse frequent and irregular, or feeble and often imperceptible. 10. Respirations short and feeble, or sometimes panting or gasping. 11. And finally, incomplete coma associated with nausea, hiccough, vertigo, dimness of vision, and painfully acute hearing.

The symptoms of the second stage (stage of *reaction*) are: 1. Slow and gradual improvement in appearance of the face. 2. Improvement in the rhythm and the force of pulse. 3. Fuller respirations. 4. Sighing. 5. Return of power of deglutition. 6. Increase of temperature. 7. Change in the position of the patient from a supine posture (sign of debility) to the

side. 8. Slight febrile action, followed by sleep, and often by convalescence.

This stage of reaction may not be uniformly progressive, but may be characterized by relapses and successive reactions. The prognosis depends on the rapidity of the stage of reaction.

The symptoms of the third stage, if present, are: 1. Dry heat of skin. 2. A rapid and bounding pulse, but always compressible. 3. Hurried but imperfect respiration. 4. Tremulous tongue. 5. Great thirst. 6. Restlessness and jactation. 7. Delirium (most marked at night). 8. Muscular twitchings.

As exhaustion comes on, the skin again becomes cold and clammy; the face pale and haggard; the pulse very rapid and fluttering; subsultus, coma, or convulsions usually precede death; death.

Treatment.—The treatment of collapse should be modified in two sets of cases. 1. Those associated with hemorrhage. 2. Those without hemorrhage.

In the first class of cases Travers makes the following suggestions as a summary of treatment. 1. Maintain a horizontal position. 2. Give brandy in *moderation* per stomach and rectum. 3. Apply hot flannels to the epigastrium and the extremities. 4. Early nourishment must be given following the use of stimulants. 5. The head should be kept cool to avoid mental excitement. 6. Early sleep, through opiates, should be obtained. Give hyoscyamus if opium fails to produce sleep. 7. If reaction be delayed beyond twelve hours, increase the stimulants, apply mustard to the epigastrium, and induce sleep through large doses of opiates.

C. T. Hunter, of Philadelphia, recommends placing patient in a bath at 98° and raising it rapidly to 110°. The respirations have thus been known to fall from 36 to 20.

Young recommends, in cases demanding serious operations, confinement of the patient, for one week previous to the operation, to the *same position* as required for the first week following the operation, in order to prevent the confinement becoming an exciting cause of shock.

In those cases not associated with hemorrhage the following treatment is suggested: 1. Opening of the ex-

ternal jugular vein, if distended, is advised, to relieve the distention of the right heart, since in experiments on animals this treatment has been proved efficacious. 2. Maintain the normal temperature of body if possible to do so. 3. Diffusible stimulants should be given with great care to avoid excessive reaction. 4. Artificial respiration should be used when demanded.

We now reach the third class of causes of death, viz.: "Causes affecting the blood."

ANÆSTHETICS.

The list of anæsthetics to day in general surgical use comprises chloroform, sulphuric ether, nitrous oxide gas, bichloride of methylene, tetra chloride of carbon.

Preparations of amyl, although anæsthetics, are not used as general surgical aids, and the various preparations employed for local anæsthesia do not enter into the cause of death, and are therefore omitted.

Effects of Inhalation.—All anæsthetics by inhalation follow, to a greater or less degree, a resemblance to the symptoms of chloroform and sulphuric ether.

In the first stage (that of *faucial* and *laryngeal irritation*) there is developed: 1. An increased flow of mucus. 2. A desire for air and sense of suffocation. 3. Frequent reflex acts of swallowing. 4. Coughing. 5. Struggles of the patient.

In the second stage (that of *general exhilaration*) we notice an increased pulse, increased respirations, flushing of the face, laughing, shouting, crying, and other symptoms of intoxication. The taste and smell are lost, and analgesia is present.

This stage is most prominent in the mercurial and hysterical temperaments.

In the third stage (the *tetanic* or *convulsive* stage), the muscles become rigid, the face cyanosed, the breathing arrested or stertorous, opisthotonos exists, and the eyes are staring and open.

This stage is least marked in women, and the feeble or debilitated.

In the fourth stage (the stage of *coma*) the surface becomes cool, diaphoresis is present, the countenance is placid, the pupils are normal or slightly contracted, the respirations become shallow but easy, and the pulse somewhat slower than normal.

CAUSES OF DEATH.

The causes of death in anæsthetics are: 1. Syncopal apnœa. 2. Epileptiform syncope. 3. Paralysis of respiratory muscles. 4. Cardiac paralysis. 5. Shock. 6. Coagulation of blood in the pulmonary capillaries. 7. Direct anæsthesia of lung tissue.

(A) SYNCOPAL APNŒA (so called by Richardson).—This cause of death occurs during the first stage of chloroform, *soon after administration of the anæsthetic is commenced.* It is explained by Richardson as due to "irritation of the peripheral nervous system associated with excess of carbonic acid in blood and arrest of heart's action," and by Bartholow, as due "to *direct* paralysis of the cardiac ganglia from contact with chloroform in the blood. These ganglia being in an abnormal state of susceptibility from causes not now understood."

(B) EPILEPTIFORM SYNCOPE.—This occurs during the *third* or *tetanic stage.* It is produced by tetanic fixation of the respiratory muscles, and interference with the pulmonary circulation, resulting in venous engorgement of right heart. Respiration in this form of death ceases before the heart's action is arrested.

(C) PARALYSIS OF THE RESPIRATORY MUSCLES.—This occurs during the stage of *complete muscular relaxation.* The heart beats for some seconds after respiration has ceased.

(D) "CARDIAC PARALYSIS."—This occurs during the stage of *complete insensibility* and analgesia. It is produced by paralysis of the cardiac motor ganglia. Respiration may continue for a short time after heart has ceased to act. J. Paget's case, 1857. Respiration seven minutes after heart failure.

(E) SHOCK.—This cause of death, when present, is induced by and combined with the respiratory or cardiac depression existing as the result of the anæsthetic. This may occur either in the early or late stages.

Faure's * theory attributes death from chloroform in all cases to coagulated or *thickened blood in pulmonary*

* Faure asserts that chloroform is never absorbed, but is always *local* in its action.

capillaries, from the direct action of too strong chloroform vapor.

Madden's * theory attributes death from chloroform to direct *anæsthesia of the lung tissue*, and therefore arrest of respiration and asphyxia.

The conditions rendering anæsthetics dangerous are: 1. Fatty degeneration of heart (a prominent contraindication). 2. Previous alcoholic history. 3. Brain tumors and degenerations. 4. Respiratory obstruction from swollen epiglottis, enlarged tonsils, œdema glottidis, laryngeal paralysis, thoracic tumors or aneurism. 5. Emphysema and obstructed pulse circulation, from engorgement of right heart, and deficient heart-power. 6. Valvular lesions, provided compensatory hypertrophy is not *proportionately* developed. 7. Incomplete anæsthesia during *painful* surgical procedures, causing death from shock, as the result of peripheral irritation.

Muscular debility and weakness from exhaustion, if otherwise uncomplicated, prove rather aids to anæsthesia than contraindications.

Preventive Measures against Danger.—These may be comprised in the following rules: 1. A thorough examination—for sources of danger should always be made previous to administration of an anæsthetic. 2. Never administer on a full stomach, as anæsthesia of the glottis prevents expulsion of vomited matter from the larynx, in case it enters by regurgitation. 3. Never administer after long *fasting*, as absence of nutrition may tend toward cardiac paralysis. 4. Give one or two ounces of whiskey before administration. 5. Nussbaum (Berlin, 1863) suggests that a hypodermic dose of morphia be given before the administration of the anæsthetic; the results which he claims being (1) that it prolongs the anæsthesia, and (2) that less anæsthetic is required.† 6. Avoid all excitement to the patient from fear, sight of instruments, too many spectators, etc., all of which tend to induce shock. 7. Have appliances for resuscitation at hand, and plenty of fresh air during the

* Tenn. Med. Society, April, 1858.

† Med. Times and Gazette, 1864; Report of Versailles Med. Society.

administration of the anæsthetic. 8. In chloroform, mix only three and a half per cent. of the vapor with air to ensure safety (Simpson's rule being ʒ ss. on a handkerchief). The specific gravity of chloroform being four times heavier than that of air, a saturated handkerchief, if held close to the mouth, will displace the air and give a dangerously large percentage of chloroform vapor. 9. In ether, the respirations alone need be watched during its administration. In chloroform, however, the respirations and the pulse need both to be carefully noted.

Treatment of Dangerous Symptoms.—1. Nélaton's plan suggests immediate inversion of the patient in case of heart failure. 2. Inhalation of gtt. v. –x. of nitrite of amyl may be given early, the tongue being drawn out to lift the epiglottis. 3. Stimulation, in case respiration is affected, but not entirely suspended, should be employed by means of either ammonia to the nostril, cold douche, or injection of ammonia into the veins. 4. Galvanism, if employed, may be administered by the following methods: (*a*) Herapath's method (*Lancet*, 1852). The positive pole is placed to the nostril and the negative pole over the diaphragm. A reflex action is thus excited between the fifth pair and the pneumogastric. This is used chiefly in case of respiratory failure. (*b*) Duchenne's method. The poles are placed directly over both phrenic nerves, on a line with the fourth cervical vertebra. This also serves to stimulate respirations. (*c*) Packard's method (*Amer. Jour. Med. Science*, 1865). One pole is placed over the upper dorsal spinous process, and the other pole over the apex of the heart. By this method cardiac contraction is induced. 5. Tracheotomy and inflation of the lungs by a catheter passed down the trachea, as suggested by Langenbeck (Berlin, 1859), may be resorted to in desperate cases. 6. Finally, acupuncture of the heart has been suggested, but as yet is not well verified as a safe procedure, nor can it conscientiously be recommended.